positive control for each sample. For the lymphocyte samples, 500 ng of each A375 LTKOSN.1 dilution of genomic DNA ($1x10^{-4}$ and $5x10^{-4}$) was used as additional controls. PCR product from blood lymphocytes and controls were transferred to membrane using a slot blot. The env probe was labeled with (^{32}P)dCTP by the random priming technique (Boehringer Mannheim). The blots were hybridized overnight at 42°C in Hybridisol (Oncor) and washed. No env gene sequence was detected by PCR in PBL samples obtained up to one year after VPC infusion.

Please replace the paragraph at page 30, beginning at line 2 with the following:

PCR primers (JMTKO1 5' TAT AGA CGG TCC TCA CGG GAT 3') SEQ ID NO: 3 and JMTKO3 5' TCA TGC TGC CCA TAA GGT AT 3') SEQ ID NO: 4 were designed to amplify a 388 bp fragment of the TK gene. The reaction mix contained 500 ng of genomic DNA sample. A375 NV cells and a sample containing no genomic DNA was used as negative controls. 100 fg of pLTKOSN.1 was used as a positive control for each sample. For the lymphocyte samples, 500 ng of each A375 LTKOSN.1 dilution of genomic DNA (10^{-2}, 10^{-3}, 10^{-4}, and 10^{-5}) was used as additional controls. 500 ng of 10^{-3} and 10^{-4} dilutions of A375 LTKOSN.1 genomic DNA were used as controls for the tumor and peritoneal wash cells. PCR product from blood lymphocytes and controls were transferred to membrane using a slot blot. A TK probe was labeled with (^{32}P)dCTP by the random priming technique (Boehringer Mannheim). PCR products from peritoneal wash and tumor samples were run out on 1.5% TBE gels and Southern transferred onto nylon membrane following manufacturer's instructions. No HSVtk gene transfer into PBL from patient blood samples up to 3 months after VPC infusion were detected by PCR in any patient.

In the Claims

Please amend claims 3, 6, 12, and 16 as follows:

3. (Amended)

The method of claim 1 wherein the subject is human and the xenogeneic cells have α(1,3) galactosyltransferase gene expression.

6. (Amended)

A method of treating tumors comprising inducing a hyperacute rejection to the cells in and/or in the vicinity of the tumor by inducing an intraperitoneal inflammatory response and thereby destroying cancer cells.